Pen 12.02 (S)
17
Copy 1

UNIVERSITY OF TORONTO MEDICAL JOURNAL

Anxiety, Personality and the Atomic Age	John K. Warnica, 5T9	32
Atherosclerosis—Recent Advances in Etiology and Pathogenesis	W. W. H. Rudd, 5T9	36
Post-Graduate Medical Training in England	Beverley Clark Burgess, M.D., 5T4	42
The Management of Benign Ano-Rectal Disease	Harvey Newton, 5T9	45
Medicine Made Easy	P. C. F. Moore, 5T9	49
Case Report	Gloria Pierce, 5T9	52
Book Review		54

DECEMBER 1958

VOLUME XXXVI NUMBER 2

FAST-ACTING ORAL BROAD-SPECTRUM THERAPY.

The modern blue and yellow ACHROMYCIN V Capsules, combining equal parts of pure crystalline ACHROMYCIN Tetracycline HCl and Citric acid, provide unsurpassed oral broad-spectrum therapy.

Speed of absorption adds new emphasis to the benefits of true broad-spectrum action, minimum side effects and wide range effectiveness that have established ACHROMYCIN as an antibiotic of choice for decisive control of infection.

REMEMBER THE V WHEN SPECIFYING ACHROMYCIN V.

New blue and yellow capsules (sodium-free)—250 mg. with 250 mg. citric acid, and 100 mg., with 100 mg. citric acid.

ACHROMYCIN V dosage: Recommended basic oral dosage is 6-7 mg. per lb. body weight per day. In acute, severe infections often encountered in infants and children, the dose should be 12 mg. per lb. body weight per day. Dosage in the average adult should be 1 Gm. divided into four 50 mg. doses.

ACHROMYCIN* V CAPSULES

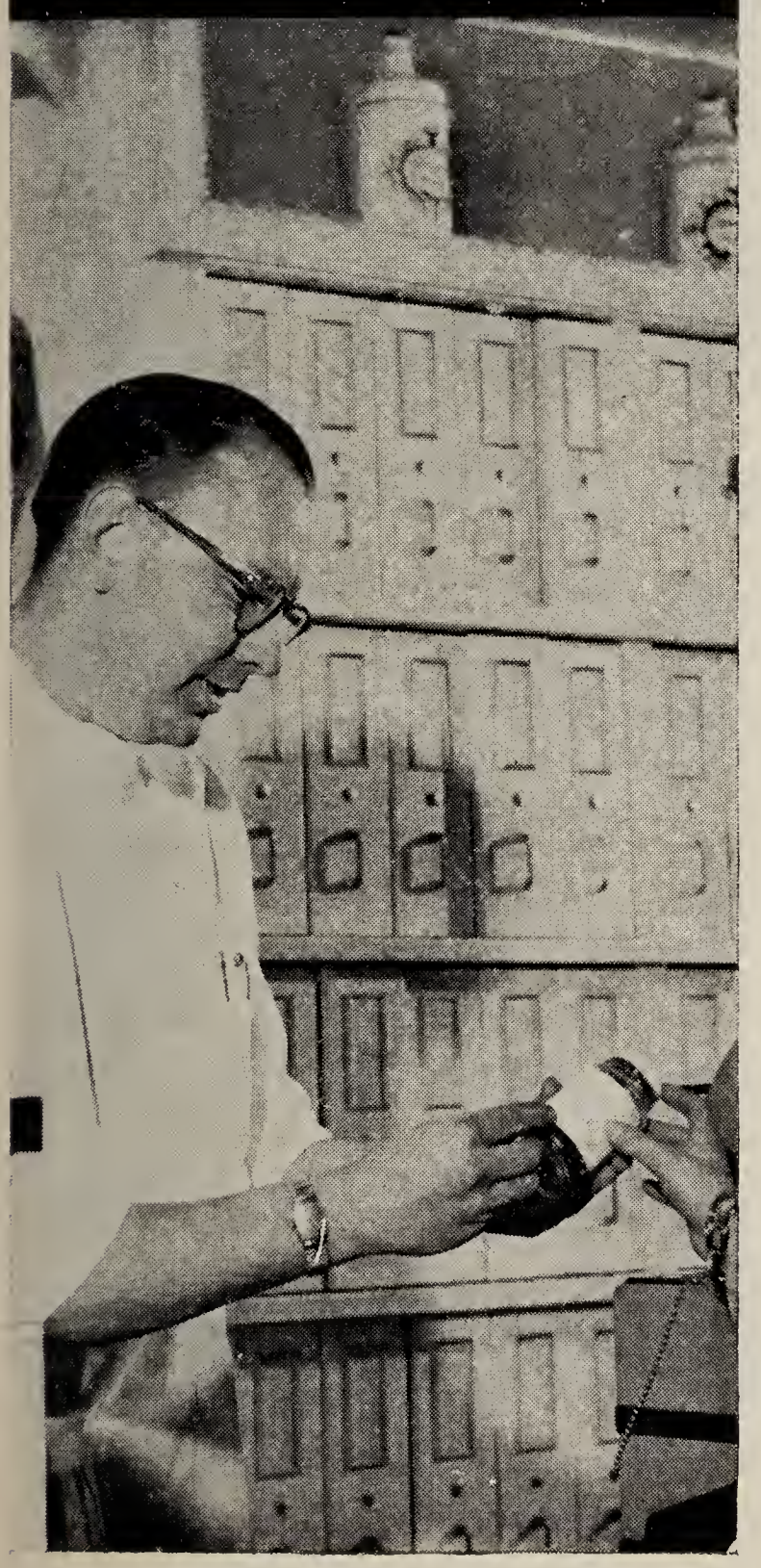
Tetracycline HCl and Citric Acid Lederle

LEDERLE LABORATORIES, a Division of CYANAMID OF CANADA LTD.
Montreal, Quebec

*Reg. Trademark in Canada

Lederle CANADA

this is your Professional Helper



. . . when the Pharmacist does ALL your dispensing

The years he has spent at his College of Pharmacy and in actual experience, qualify him to render you expert assistance in all things pharmaceutical, leaving you more time for enlarging your practice, for studying or for leisure.

ONTARIO RETAIL PHARMACISTS' ASSOCIATION

THE ONTARIO RETAIL PHARMACISTS' ASSOCIATION

Baby's Own Tablets

satisfactorily relieved

every one of 40 babies* with

constipation

and 34 out of 35 babies* with

teething

gastrointestinal upset and malaise

with complete easing of straining at stool, gas distress, disturbed sleep, restlessness, crankiness and anorexia.

REMARKABLY SAFE — "Throughout the study . . . in no instance was there any untoward reaction" whatsoever.

BABY'S OWN TABLETS provide Phenolphthalein 3/16 grain, mildly buffered with Precipitated Calcium Carbonate ½ grain, and Powdered Sugar q.s. Pleasant, convenient.

*2 months to 24 months of age.

For a sample supply and literature citing references 1-15 write . . .

Typical Case History

CASE #50. Baby R.S., age 12 months, weight 20 lb. 10 oz., had gastrointestinal discomfort and malaise associated with teething. Baby had no teeth as yet, but gums were tender, puffy and swollen. Baby was cranky, irritable, restless and couldn't sleep. Drooling was excessive; appetite poor.

BABY'S OWN TABLETS were given, one each night at bedtime.

Baby had satisfactory relief of symptoms. Appetite improved. First days, then nights, became more comfortable. Baby now has six teeth.

G. T. FULFORD CO., LIMITED, Brockville, Ontario

Elastoplast
TRADE MARK

THE **POROUS** ADHESIVE

Years of extensive clinical trial and successful use in Great Britain and Canada have shown that *only* Elastoplast Porous Adhesive provides all these advantages:

- Adequate Porosity throughout the entire surface of the adhesive that permits free sweat evaporation and reduces skin reaction.
- The proper degree of Stretch and Regain for correct compression and support.
- Fluffy edges to prevent trauma and devitalized skin.

Elastoplast *The synonym for quality and reliability in the surgical field*

S&N **SMITH & NEPHEW, LIMITED**

5640 Paré Street, Montreal 9, Que.

announcing

a new topically superior

now available

for topical application in dermatotherapy

Kenalog (triamcinolone acetonide) is a new synthetic corticoid compound with powerful anti-inflammatory and antipruritic action. Developed by the Squibb Institute for Medical Research, and evaluated during preliminary clinical trials in over 1,000 patients, Kenalog used topically has demonstrated its greater effectiveness in controlling most common dermatoses.[1] Symptoms of itching and burning are dramatically relieved after topical application of Kenalog.[1,2] A superior agent for both acute and chronic dermatoses, its greater anti-inflammatory action is most clearly apparent in the treatment of chronic, therapeutically refractory conditions, such as chronic eczematous dermatitis.[3] Complete resolution is often obtained with Kenalog where other topically applied steroids have failed.[3]

Kenalog in ointment, cream or lotion form may be used for treatment of a wide variety of dermatoses including:

Atopic dermatitis
Contact dermatitis
Eczematous dermatitis
Neurodermatitis
Seborrheic dermatitis
Insect bites
Pruritus ani
Pruritus vulvae
Lichen simplex chronicus
Exfoliative dermatitis
Stasis dermatitis
Nummular eczema

Kena

RELIABILITY UNIFORMITY PURITY EFFICACY SQUIBB

SQUIBB

A Century of Experience Builds Faith

anti-inflammatory steroid

Kenalog safely and effectively provides for prompt symptomatic relief as well as control of many common dermatologic disorders.[1,7]

In double-blind tests in 309 patients comparing 0.1% Kenalog and 1.0% hydrocortisone, Kenalog exhibited superior anti-inflammatory, antiallergic and antipruritic activity.

Investigator	Total Cases	Kenalog Superior	Hydro-cortisone Superior	Neither Effective	Equally Effective
Goodman[3]	50	32	3		15
Smith et al.[4]	109	75	3	3	28
Fitzpatrick et al.[5]	120	61	5		54
Lerner[6]	30	20	4	1	5
	309	188	15	4	102

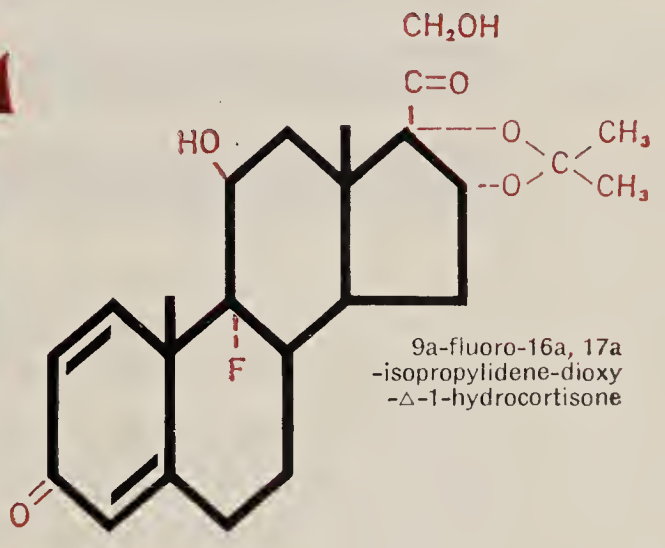
Kenalog is extremely well tolerated locally. No systemic toxicity has been observed in clinical studies, published and in progress, involving over 1,000 patients.[1,3] Metabolic studies show that there is no electrolyte disturbance when Kenalog is applied topically.[1,4,5]

1. Reports to the Squibb Institute for Medical Research.
2. Howell, C. M.: Squibb Clinical Research Notes 1:5 (Oct.) 1958.
3. Goodman, J. J.: Ibid. p. 1.
4. Smith, J. G.· Zawisza, R. J., and Blank, H.: Ibid. p. 6.
5. Fitzpatrick, T.; Crowe, F. W., and Walker, S. A.: Ibid. p. 12.
6. Lerner, A. B.: Ibid. p. 2.
7. Robinson, R. C. V.: Bull. School of Med., U. Maryland 43:54 (July) 1958.

Supply
Kenalog cream, 0.1% — 5 Gm. tubes.
Kenalog ointment, 0.1% — 5 Gm. tubes.
Kenalog lotion, 0.1% — 15 cc. plastic squeeze bottles.
Kenalog-S lotion, 0.1% — 15 cc. plastic squeeze bottles.

log

Triamcinolone Acetonide



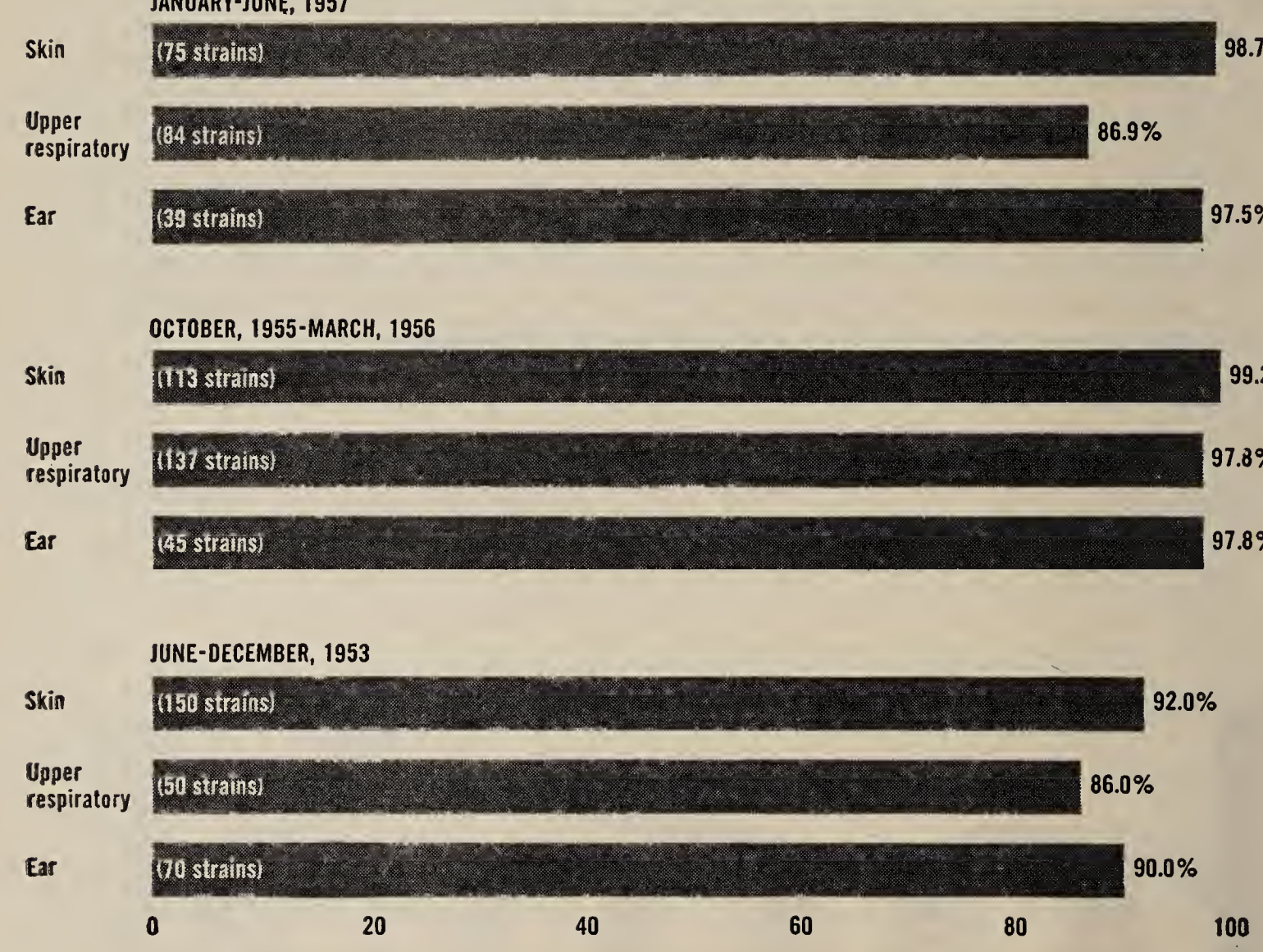
9a-fluoro-16a, 17a
-isopropylidene-dioxy
-△-1-hydrocortisone

KENALOG IS A SQUIBB TRADEMARK

HIGHLY EFFECTIVE AGAINST STAPHYLOCOCCI ...YEAR AFTER YEAR

CHLOROMYCETIN®

IN VITRO SENSITIVITY OF STAPHYLOCOCCI FROM THREE FOCI OF INFECTION TO CHLOROMYCETIN FROM 1953 TO 1957*



*Adapted from Royer, A., in Welch, H., & Martí-Ibañez, F.: Antibiotics Annual 1957-1958, New York, Medical Encyclopedia, Inc., 1958, p. 783.

CHLOROMYCETIN (chloramphenicol, Parke-Davis) is available in a variety of forms, including Kapseals® of 250 mg., bottles of 16 and 100. CHLOROMYCETIN is a potent therapeutic agent and, because certain blood dyscrasias have been associated with its administration, it should not be used indiscriminately or for minor infections. Furthermore, as with certain other drugs, adequate blood studies should be made when the patient requires prolonged or intermittent therapy.

PARKE, DAVIS & CO., LTD. · TORONTO 14, ONTARIO

MEDICAMENTA VERA

46558

UNIVERSITY OF TORONTO
MEDICAL JOURNAL

ESTABLISHED DECEMBER 1923
AN UNDERGRADUATE PUBLICATION

VOLUME XXXVI NUMBER 2

TORONTO, DECEMBER, 1958

Editor-in-Chief ..... P. C. F. Moore, 5T9

Associate Editor ..... P. B. Dent, 6T0

Assistant Editor ..... R. F. Armstrong, 5T9

Business Manager ..... J. R. Lundon, 6T0

Advertising Manager ..... R. D. J. Brand, 6T2

Assistant Advertising Manager ..... Miss J. Shkimba, 6T2

Circulation Manager ..... G. V. Boujoff, 6T2

This Journal is published six times a year from November to April, by the Medical Society of the University of Toronto, to serve primarily as a medium for student expression.

Subscription rates: $3.50 for one volume of six consecutive issues. Single copies 65¢.

Manuscripts including references and footnotes should be typewritten double spaced on one side of the paper. References should be kept to a minimum, and should be cited numerically in the order in which they appear in the text.

Manuscripts in excess of 3,000 words will be considered *unsatisfactory.*

Illustrations are included by arrangement with the Editor. Drawings should be done on white paper in black ink.

Final copy date is the first day of *two months* prior to publication, e.g., September 1 for the November issue.

Material printed in this Journal is covered by copyright. Papers are accepted for consideration with the understanding that they are contributed for publication in this Journal only.

Correspondence should be addressed to Room 114, Medical Building, University of Toronto, Toronto 5, Ontario.

Authorized as Second Class Mail, Post Office Department, Ottawa.

ANXIETY, PERSONALITY AND THE ATOMIC AGE

JOHN K. WARNICA, 5T9

> "There is no cure for the body apart from the mind. First, then, and above all, the mind must be treated if the body is ever to be made whole . . ."
> —Plato, *Phaedo.*

TO discuss a topic of such shocking magnitude, and to attempt to organize and reduce one's comments into the neat nut-shell of a few pages is an exceedingly difficult task, and is, perhaps, a foolhardy undertaking. The terms used in the title of our discussion, while at first appearing grand and pontifical, and though they slide easily from the slick tongue of the popular press, are, nonetheless, poorly defined in the thinking of most people. This difficulty is compounded by the annoying fact that various authors employ these terms in different ways; they may be used in a very restricted sense by one man, and in a very unrestricted way by another. As well, it is difficult for the investigator to correlate the literature which deals with our topic; difficult to weigh the conclusions of the sociologist against the views of the psychiatrist; and difficult to equate and measure the stresses of our time with those of past centuries.

Ultimately as one sets out to write on this topic, reference texts and statistical analyses must be put aside and one must boldly set forth a discussion which culminates from a collection of personal impressions. We acknowledge that this report may lack the authenticity of scientific fact. We realize that the nutshell of space precludes extensive elaboration. We recognize that our conclusions will probably be sweeping and oversimplified. Yet we hope that this discussion will provide the inquirer with a basic understanding of neurotic anxiety and how it afflicts the personality of Man as he faces the complex stresses of the Atomic Age.

Anxiety

The term *anxiety* used in its clinical sense, can be defined as an emotional state of fearful apprehension, associated with the physiological concomitants of fear. It is important to note that anxiety has a subjective component—"the fearful apprehension", as well as an objective component—"the physiological concomitants." Therefore in recognizing anxiety as it underlies the psychoneurosis, the signs, as well as the symptoms of anxiety must be observed before its presence can be confidently recorded.

The etiology of anxiety is stress. Stress nibbles and gnaws on the corpus of personality, causing psychic pain or anxiety to appear. Unconsciously the neurotically anxious individual allows insidious, unfounded fears to develop, and he begins to walk upon the hot coals of continuous apprehension. His attempts to reduce his anxiety are usually inadequate, unrealistic and immature. Instead of facing his anxiety squarely, he attempts to bury it in his thinking, or conceal it by somatic symptomatology, or by salving over the wounds of anxiety by alcohol or drug addiction, or by hiding his apprehensions with some other disguise. In this way a self-perpetuating, and indeed, a self-potentiating cycle becomes established.

Cannon, Selye and others have attempted to explain this phenomenon on a physiological basis. Stresses trigger off central nervous system centres, probably located in the hypothalmus, to send

nervous impulses to the adrenal medulla, causing the consequent liberation of adrenalin into the blood stream, which in turn produces an imbalance of the autonomic nervous system. Due to the hyperactivity of the sympathetic system, the physiological manifestations of anxiety result. Applying this concept to the psychoneuroses, in the *acute anxiety state* one sees the signs and symptoms of acute fear. Initially the patient has a premonition of imminent disaster; this is rapidly followed by a fearful agitated facies, with dilated pupils, hyperventilation, trembling, perspiration, palpitations and giddiness. In the *chronic anxiety state* one notes the vague but liturgical symptomatology of nervousness, breathlessness, palpitations, easy fatiguability, dizziness, headaches, trembling, chest pain, epigastric fullness, "fluttering" abdominal distress, constipation, anorexia, frequency and paraesthesiae. Physical examination may reveal a slight tachycardia, flushing, tremor, bilaterally hyperactive reflexes and exaggerated reaction to painful stimuli.

The relationship between anxiety and mental illness—particularly psychoneurotic illness—appears to be unrecognized by some students. Many investigators have asserted that anxiety is the dominant etiological force at work in the production of the psychoneuroses. Anxiety may boldly appear in an anxiety state *per se* (*vide supra*), or may insidiously underlie hysteria, obsessive-compulsive states, reactive depressions or phobias. Indeed, Freud stated that anxiety was "the fundamental phenomenon and the central problem of the neuroses". Viewed in this way, the manifestations of neurotic anxiety, altered by the patient's age and sex and temperament, and moulded by a grotesque variety of internal and external stresses may present as any one of the psychoneurotic diseases.

Personality

A brief scrutiny of personality will now be undertaken, for it will help the reader to comprehend with greater clarity the mental mechanisms under discussion.

Over the past century, a great many theories have been propounded in an attempt to explain personality. None of these theories is entirely satisfactory. However, Freud's concept concerning the structure of personality is the most widely accepted in clinical medicine and we shall undertake to discuss it briefly.

Freud felt that personality was a composite of three major systems, the *id*, the *superego* and the *ego*. While each system has individual properties, together they interact smoothly in the mentally healthy individual so that it is impossible to distinguish clearly the boundaries of each system within that personality.

The id is the basic, passionate stuff of the personality—the rough uncut diamond of Man, with instinctual behaviour, libido and self-preservation being facets of this jewel. Id resides in the "unreal" internal world of man's unconsciousness.

The superego is the conscience of the personality, the policeman, schooled in the traditions and morals of our culture by parental guidance; superego strives for the perfection of the personality as it censors the libidinous psychic energy of the id.

The ego is that part of personality which relates the internal world of the individual to the external world about him. Ego plays the role of an executive, attempting to integrate the often conflicting demands of the id and the superego to the external world.

As Hall and Lindzey point out, "in a very general way, the id may be thought of as the biological component of personality, the superego as the social component, and the ego as the psychological component".

While the external or "real" world

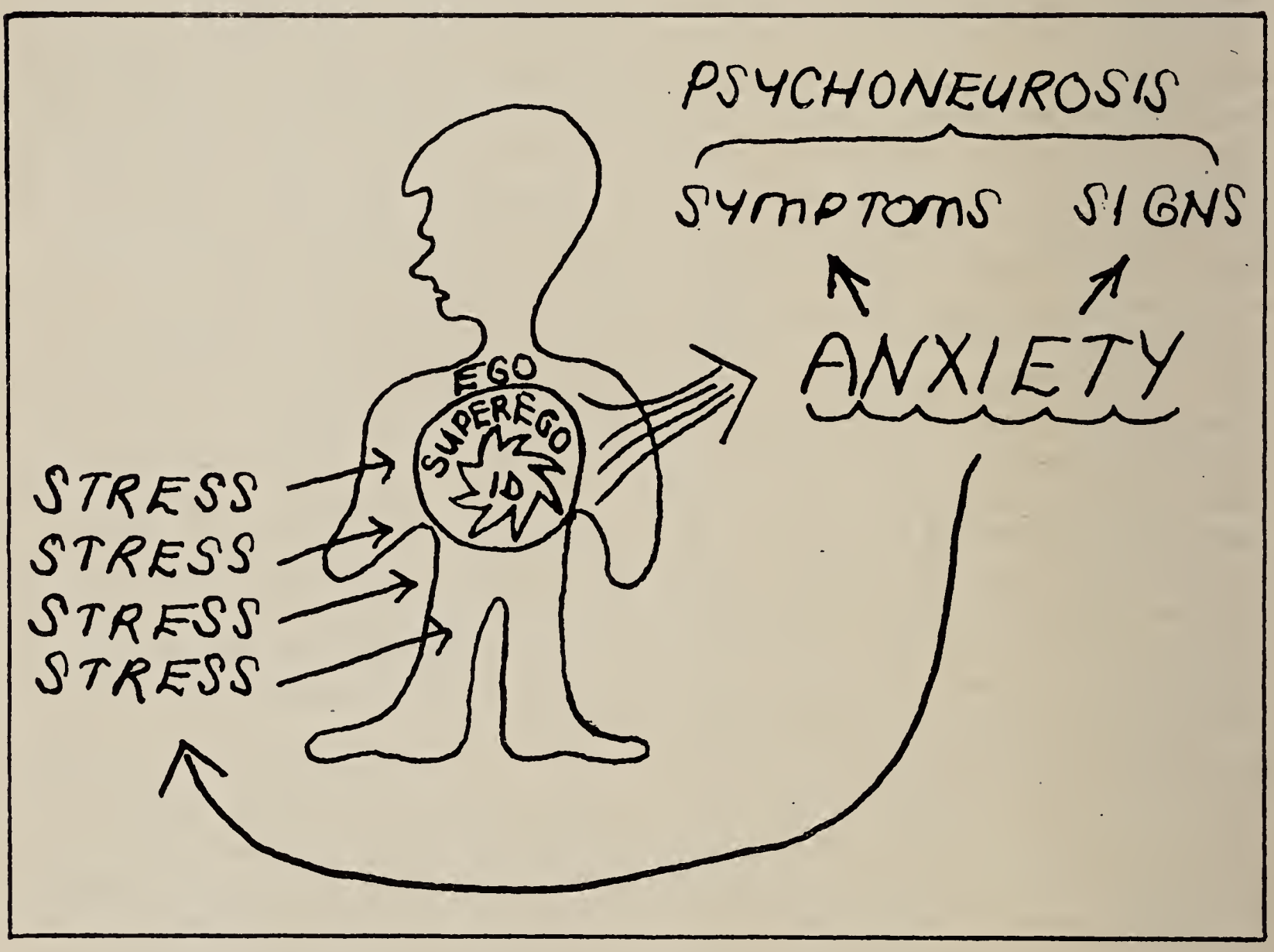
provides pleasure, and therefore allows ways by which the internal tensions may be relieved, yet our environment also contains threatening forces or stresses which increase internal tensions.

In the neurotic individual these stresses may assume Brobdingnagian proportions, the id threatens to erupt through the cordon of the superego, and the ego becomes flooded with anxiety.

The foregoing discussion may be simply summarized diagrammatically:

. . . And The Atomic Age

It is difficult to prove, but it is the popular impression that anxiety is increasing in the Atomic Age. One can measure anxiety indirectly by analysing neurotic stress-reducing reactions, such as alcohol or drug addiction, and note that there is a recent real per capita increase in these vices. Yet one cannot titrate anxiety directly and relate it quantitatively to its presence a century ago. Commonly heard, wistfully uttered



PERSONALITY OF MAN is shown divided into three major systems: internally in our unconsciousness is found the id, the rough, passionate core of man, encircled by the censoring activity of the moralistic superego. Ego attempts to control the resultant internal tensions, and to relate the personality to the external world.

External stresses assail the personality and add to pre-existing internal stresses. In the psychoneurotic personality, anxiety floods the ego. Due to faulty reactions to anxiety, a self-perpetuating, self-potentiating cycle is established, leading to frank psychoneurotic anxiety. This apprehension may show itself in the varied symptomatology of any of the psychoneuroses.

remarks such as, "Oh for the good old days," or "What is the world coming to?" befog the issue, making accurate appraisal more difficult.

Yet it may be suspected that there are two mental mechanisms at work in this age which tend towards the production of a tense psychoneurotic 20th-Century Homo Sapiens.

The first of these mechanisms concerns itself with the impression that our culture breeds a crop of lily-livered, luxury-laden, immature adults who, living in their honey-coated world never learn to face reality until they are finally jettisoned into the ruthless battlefield of business, into the sobering facts of marriage, or into the self-conscious sphere of society. The stresses of reality suddenly thrust upon them are greatly magnified; and ill-equipped to wrestle with these challenges, Atomic Age Man apprehensively reacts by yearning for security.

The second mechanism deals with man's rapid technical advancement during the atomic age. The geni, dressed in the guise of the Machine, is a new and ominous stress. Man has harnessed the machine to meet his daily needs—to awaken him, to shave him, to prepare his food, to carry him to work and home again and then to entertain him for the evening. The harnessed machine is a very willing, capable and efficient servant, answering man's every whim.

On the other hand, the machine frightens as well as pleases. The temperamental mechanical brain doted upon by skilled mathematicians can rapidly solve mathematical, economic and scientific problems. Its I.Q. easily eclipses man's puny brain. Prophets concerned with the science of cybernetics visualize a future filled with machines energized by electrical central nervous systems, solving and legislating our social problems. An unhappy prospect indeed, man mastered by his offspring.

And yet such a time might well never arrive—the offspring may annihilate the master. At present we suspect that a large proportion of the world's population is but a push-button's distance from atomic slaughter; and if not slaughter, then at least radiation disease—fearful burns, hideous genetic monsters, impotence, cancer.

Atomic Age Man falls back aghast, like Pandora, terrified at the forces he has unleashed; and his fears, often ill-founded upon mystery, superstition or ignorance, give way to neurotic anxiety.

Summary

Anxiety is not foreign to our time. History tells us that neurotic anxiety has always hampered man's effectiveness. But what we do suspect is that there has been a recent increase in psychoneurotic anxiety, due to man's greater insecurity, and therefore, greater inability to handle the increased complex stresses that are his heritage in the Atomic Age.

Bibliography:

1. Adams and Hope: Anxiety State and Psychaesthenia. Harrison: Principles of Internal Medicine; 1958, Ed. 3.
2. Curran and Partridge: Psychological Medicine, 1957, Ed. 4.
3. Henderson and Gillespie: Textbook of Psychiatry, 1955, Ed. 7.
4. Hall and Lindzey, Theories of Personality, 1957, Ed. 1.
5. Stern: Third Revolution, 1954, Ed. 1.
6. Meerloo: Patterns of Panic.
7. Weiner: Cybernetics.

ATHEROSCLEROSIS—Recent Advances in Etiology and Pathogenesis

W. W. H. RUDD, 5T9

A great deal of attention has in recent years been attracted to the problem of atherosclerosis because it is conceded to be the only important lesion which produces coronary stenosis or occlusion. Observers have been further stimulated by the real or apparent great increase in the morbidity and mortality from coronary artery disease. For many years blood cholesterol levels and dietary factors leading to hypercholesterolemia have been the prime concern of those searching for further knowledge in the etiology and pathogenesis of atherosclerosis. In the light of the recent work of several investigators, evidence has been accumulating to suggest that a fundamentally new approach to the problem may now be necessary.

If a theory, whether new or old, is to be acceptable, it must explain certain acknowedged facts. It might therefore be of advantage to review first some of the more important of these.

1. Atherosclerosis is extremely common. Early signs of the characteristic lesions may even be found in children. In a series of three hundred American soldiers killed during the Korean war, having an average age of 22 years, there was gross evidence of coronary atherosclerosis in 77%[1].
2. The distribution of the lesions must be explained i.e.: the sites most often and severely affected are the flexions, bifurcations and branchings. Arteries that are poorly supported by surrounding tissues are also affected, for example, the extramural coronary arteries, the cerebral arteries and arteries of the circle of Willis.
3. The theory must take into consideration that the incidence and severity of the process is more marked in the hypertensive individual.
4. It is of interest that man, the chick and the duck are predisposed to atherosclerosis and the lesion is not recognized in any other animals[2]. Why is this?
5. The increased incidence in obese patients and in certain disorders such as hypercholesterolemia, diabetes, nephrosis, hypothyroidism and familial xanthomatosis which lead to atherosclerosis at an earlier age and to a more severe degree, should be accounted for.
6. The incidence before the age of thirty in males versus females is two to one. This ratio decreases towards the menopause and thereafter tends towards a ratio of one. Thus a hormonal factor is implicated in the atherosclerotic process and needs to be explained by the theory.
7. Lastly, the presence of the typical cholesterol crystals, the phospholipids, fatty acids and blood pigments in the atheromatous plaque must be taken into consideration.

Certain histological and physiological peculiarities of the blood vessels which are important to the explanation of the etiology and pathogenesis of the disease might first be recalled. Of the three coats of the blood vessel wall, the tunica intima of the larger elastic arteries warrants our detailed consideration. The intima constitutes about one-sixth of the thickness of the wall. It may be divided into four parts. Its lining consists of a smooth

layer of thin endothelial cells. Outside this is the subendothelial layer of the intima which consists of delicate elastic fibres together with some fibrous or collagenic fibres which are supported in a mucinous ground substance (or amorphous intercellular substance). This rests on the deep or external layer whose elastic fibres are somewhat coarser than those found in the subendothelial layer. Finally the elastic fibres are condensed to form an internal elastic lamina which abuts against the media. It is of interest to note that the outer three layers described are extremely thin at birth and increase in width with age. In the light of our present knowledge, the main blood supply to the arterial wall probably originates from the vasa vasorum which penetrate only as far as the media of the vessel. Similarly the lymphatic drainage penetrates only as far as the outer portion of the media. Thus the blood and lymph supply of the vessel wall is incomplete and might well present unique problems of nourishment and drainage.

Certain physiological functions of the arteries which are of fundamental importance to the explanation and reasoning of the new hypothesis must now be presented. As a result of systolic ejection of blood from the left ventricle, the aorta and its main branches dilate and later recoil in an effort to accommodate the increased volume of blood. This dilatation and recoil is repeated with each pulse beat and is accentuated in hypertension. Thus we have both the systolic and diastolic pressures. This pulse pressure difference is gradually decreased as blood flows to the smaller vessels and eventually becomes a nearly even stream.

Obviously therefore, the larger vessels are under the greater stress. It has also been determined that the greater the diameter, the greater the stretching of the arterial wall. Further, it is obvious that greater stress would be present where the aorta or vessel bends, divides or branches, and would be accentuated in arteries which have less external support and therefore greater movement. Lastly, it is important to realize that the arterial wall is made up of three fundamentally differently tissues: muscle, elastic and fibrous tissue. Each of these differs in its elastic properties, so that it is now thought that most of the post-systolic recoil of the arterial wall is due to the elastic recoil of the muscle fibres of the media, somewhat less to the stretched elastic fibres, and little if any to the fibrous tissue. Therefore intrinsic stress is present within the normal wall, but to a much greater extent in the area of a fibrous atheromatous plaque.

The Mechanical Hypothesis

As early as the year 1926, Duguid suggested the possibility that it was the impairment of expansion and recoil movements of the intima of the larger vessels due to fibrous thickenings which led to the atherosclerotic lesions. Recently Duguid and Robertson[3] have advanced further evidence in support of the mechanical theory.

The characteristic dense fibrous plaques of atherosclerosis which are found in the intima of the larger elastic vessels could be likened to an oval plate replacing part of the innermost layer of a laminated soft rubber tube closed at one end and attached to a large syringe filled with fluid at the other end. When the fluid is passed into and out of the soft rubber tube, the walls expand and contract under the pressure but the attached metal plate cannot. Thus great stress exists between the plaque and its surrounding but pliable tissues so that eventually the media and the fibrous plaque may be split apart. However, unlike the metal plate, the fibrous plaque, before it becomes consolidated, is

stretched by repeated systolic extension of the vessel wall and becomes firm in the expanded position of systole. This is the explanation for the post mortem observation that atherosclerotic vessels are in reality larger than normal ones. Thus during diastolic recoil of the vessel wall, the fibrous intima may be torn from the media and forced into the lumen. One might ask why there are not more vascular occlusions due to this phenomenon. The answer would be twofold: firstly, that the atherosclerotic arteries naturally become wider, and secondly, that the blood stream tends to beat the intimal bulging back against the wall. However, it is doubtful that in life the fibrous intimal bulging occurs with as great a frequency or to as great an extent as we are accustomed to seeing at autopsy, since the absence of the fluid pressure of the blood from within allows much greater contraction of the vessel wall at post mortem.

Thus the Mechanical Hypothesis deals with the development of the lesion after the fibrous plaque has been formed in the intima, but how do we explain the appearance of this earlier stage?

Thrombogenic Hypothesis

As far back as 1946 and 1948, Duguid investigated cases of coronary and aortic atherosclerosis for evidence of mural thrombosis and came up with some remarkable findings. Thrombi could be found in a large number of the cases and were of all sizes ranging from large masses of clotted red cells down to minute deposits of fibrin. These observations have been confirmed by Harrison[4] where particles of fibrin were injected into the veins of rabbits and a typical lesion was found in the smaller pulmonary arteries of the animal; by Crawford & Levene[5], by Movat of our own Department of Pathology, and by Prior and Hutter[6] in their mechanical needle traumatization of the abdominal aorta of rabbits. Thus when the thrombus adheres to the wall of an artery, the endothelium disappears and the thrombus becomes organized by fibrous tissue and is engulfed in the vessel wall. It has often been shown that the surrounding endothelium grows over the thrombus and once again becomes continuous. The thrombus is then beaten flat by the blood pulsation unless it is large and then some hindrance to the blood flow will result. The fibrous organization continues until a plaque is formed which becomes firm and fixed in the systolic dilated position of the vessel wall as described above. At this point the Thrombogenic Hypothesis leads directly into the Mechanical one.

Most observers agree with Duguid that thrombi are very frequently found associated with atherosclerotic plaques. However, they are somewhat more reluctant to accept his hypothesis that the whole lesion is initiated by the deposition of thrombi and their incorporation into the intima. Duguid further suggests that the continual deposition of fibrin and its associated reaction may be the explanation for the fact that the subendothelial and deep layers of intima, which are very thin at birth, become increasingly thicker with age, containing a greater proportion of collagen within the mucinous ground substance of the intima. Thus there is little difference between the process of normal physiological thickening of the intima with age and the abnormal pathological process which also becomes more marked with age. Most observers will only go so far as to say that mural thrombosis is an important factor in the growth of the atherosclerotic lesion. However, certain electron microscope determinations, carried out by Levene[7], who found fibrin to be the main fibrillary constituent of the plaque, adds weight to Duguid's hypothesis. Whatever function thrombosis plays, it is

certain that it plays an important part on the pathogenesis, since repeated mural thrombi cause the characteristic laminated appearance of the plaque where one layer of collagen has been formed on top of another. Thus either way, it is the current feeling of many that the most promising field for further investigation, for both experimental and therapeutic reasons, is the problem of thrombus formation and deposition.

The importance of thrombus formation and deposition in further stimulating the growth of an already present atherosclerotic lesion has been accepted without question; it is also considered likely in physiological thickening and the pathological initiation of a plaque. As discussed earlier, the atherosclerotic lesion itself does not cause narrowing of the arterial lumen, but rather widening. It is thought that the repeated engulfment of mural thrombi is the mechanism of gradual arterial occlusion, regardless of how the atherosclerotic lesion is initiated, and it has been suggested that the thrombi causing normal reparative arterial thickening, and those causing atherosclerotic lesions and pathological narrowing may be quite different in nature and/or size.

Blood Coagulation and Diet

The factors in the blood and tissues which control thrombus formation and keep it within physiological limits may fail. Thus the equilibrium, which Astrup, (1956), postulated as the removal of circulating fibrin by fibrinolysin, may be upset. Interestingly enough, recent work has linked up thrombus formation and diet. It has been known for some time that following a meal rich in cholesterol and fats there is an increase in the blood coagulability. In 1956 O'Brien[8] implicated the phospholipid content of the fatty meal as the important factor causing increased coagulation. Other investigators[9] found in *in vitro* brain tissue studies, that two phospholipid materials were active in the coagulation equilibrium, one acting as an accelerator and the other as an inhibitor. It was later found that this phospholipid coagulation accelerator was present in higher concentration in milk and milk products than in other fat-containing foods. Current investigation at this centre and elsewhere should soon throw more light on this problem.

We have presented the mechanical and thrombotic hypotheses and have suggested how they may be related to blood clotting factors and these to dietary factors. As suggested earlier, we might discuss now how well these associated theories explain some of the commonly observed factors of atherosclerosis that were noted above. This is done with the hope that it may prove to be stimulating and with no attempt at being complete. They will be discussed in the same order and will serve to summarize many of the points made above in a more practical light.

1. The fact that atherosclerotic lesions are so extremely common and are so frequently associated with medical thinning and tearing of the elastic lamina are strong points in favour of the mechanical theory of Duguid which would also more than adequately account for the increased incidence and severity with age.
2. The sites where plaques are most severe, on closer examination, are found to be those where the hydrostatic pressure, the degree of expansion, and the extent of vessel movement are the greatest. Thus it may be the added factor of mechanical stress that determines the site where lesions occur, but more likely it is for this reason that otherwise

physiological thickening becomes a pathological lesion.

3. Hypertension will exaggerate all the effects of mechanical stress enumerated above and is a strong point in favour of the mechanical theory.
4. That only man and very few other animals are affected by atherosclerosis is more difficult to account for. The problem here becomes very involved and may have something to do with serum phospholipid concentrations and ratios apparently unique to man.
5. The increased incidence in obese patients and those with hypercholesterolemia etc., would be more easily explained by the dietary hypothesis but here again statistical evidence previously used to correlate the two has been largely proved wanting. Indeed Yudkin[10] in his extensive statistical analysis has claimed that the incidence of coronary thrombosis in England is closely correlated to the number of radio and television licenses sold; another correlation was with the number of registered automobiles. However, it is quite likely that these metabolic disturbances cause altered blood coagulability states which favour deposition of thrombi and hence either the initiation of, or certainly the acceleration of the atherosclerotic process.
6. The relative immunity of pre-menopausal women to atherosclerosis as compared to men of the same age, together with the finding of diminished atherosclerosis in males with high blood estrogen levels secondary to liver disease and in those on estrogen therapy for prostatic carcinoma, suggests that the female sex hormone plays an important protective role. It has been shown by Russ, Eder & Barr[11] that administration of estrogens to atherosclerotic patients corrects the abnormal lipoprotein pattern in the plasma, while androgen administration worsens it.
7. Finally how do these theories explain the presence of the characteristic fatty contents of the plaque?

Duguid[3] suggests that these contents may be derived from two sources.

First it has been noted by several observers that certain types of thrombus, especially the red cell type, may undergo softening and fatty degeneration. This softening and fatty degeneration provides a very poor foundation for fibrous tissue organization to build on and thus remains unchanged. However, this method can only account for a small part of the collection of fat and this in only the small plaques, and therefore the following method is the more important. This is that the contents of the plaque are derived from the blood stream directly. Duguid claims evidence for this phenomenon in that the concentrations of lipids, cholesterol etc., found in the plaque, closely parallel their concentrations in the blood, taking into consideration the fact that some are selectively removed. There are two difficulties with this explanation. One is that vessels cannot always be found communicating with a plaque and secondly that microscopically, red cells or thrombi cannot be found in the smaller plaques. No problem is presented by the larger plaques which are supplied by a vascular ingrowth from the vasa vasorum. However, in those plaques where no arterial communication was found, hemorrhage was often present. Patterson *et al.*[12] found that iron pigment is demonstrable by chemical means even in the very early and small lesions. Duguid points out that a possible route for the attraction of blood through the vessel wall from the lumen is present during recoil of the vessel wall

around a fibrous plaque, where a negative pressure is created within the tear. This negative pressure may very well draw contents of the blood into the tear. The finding of hemorrhage associated with at least 66% of the atherosclerotic lesions and the microscopic distribution of these hemorrhages when the bleeding is minimal, (that is, located at the margins of the tear), are held as evidence by Duguid of the mechanical stress associated with the fibrous plaque. He believes that the cholesterol crystals, phospholipids, fatty acids etc., are brought to the mechanically formed lesions by the blood stream, and that they are just markers of the previously formed lesion. He suggests that abnormal blood lipid levels are therefore of much less importance than previously thought. However, the possibility that the accumulation of cholesterol etc. in the plaque may cause a foreign-body type of reaction, and hence increase the severity of the lesion, is quite likely, and must be borne in mind. Further support has been given in the campaign against the acceptance of a single dietary factor as the cause of atherosclerosis and vascular thrombosis by John Yudkin[10]. Thus, although far from being proven yet, Duguid's explanation has gained many followers and is in accord with the findings of others.

Conclusion

Although the two main theories of the etiology of atherosclerosis would appear to be completely opposite in nature, recent evidence has suggested that the same type of diet as that advocated by enthusiasts of the dietary hypothesis causes increased blood coagulability and hence thrombin deposition. The feeling is that the most promising field for further research is in the area of blood coagulation mechanisms, but because this is such a vast field in itself, this topic deserves a paper of its own. It was suggested that it was possible that mural thrombi do initiate the process of atherosclerosis, and although this is still controversial, nevertheless, it is widely accepted that thrombi are important factors in the pathogenesis. The way in which so many observed facts were so well explained by the mechanical stress theory recently revived and clarified by Duguid was described, and the fact that recent evidence in support of this hypothesis has been uncovered during research into the thrombotic hypothesis was mentioned. Certain weaknesses in accounting for some known facts by the theory were pointed out. The general trend of interest in two dietary phospholipid materials which have just recently been shown to have accelerator and inhibitor effects on blood coagulation respectively will now likely yield the most fruitful results and would help complete the gaps remaining in the knowledge of the etiology and pathogenesis of atherosclerosis.

Bibliography:

1. Robbins, S. L.: Textbook of Pathology, W. B. Saunders Co., 1957, pp. 505–514.
2. Price, D. C.: U. of T. Medical Journal 1957, IV, pp. 94–99.
3. Duguid, J. B. & Robertson, W. B.: Lancet 1957, June 15, pp. 1205–1209.
4. Harrison, C. V.: J. Path. & Bact., 1948, 60, pp. 289–293.
5. Crawford, T. & Levene, C. I.: J. Path. & Bact., 1952, 64, pp.523–528.
6. Prior, J. T. & Hunter, R. V. P.: Amer. J. of Path., 1955 XXXI No. 1, pp. 107–123.
7. Levene, C. I.: Lancet, 1955, Path. p. 1216.
8. O'Brian, J. R.: Lancet 1956 (ii) p. 232.
9. Barkham, Newlands & Wild: Lancet 1956 (ii), p. 234.
10. Yudkin, J.: Lancet, 1957, July 27, pp. 155–162.
11. Russ, Eder, Barr: Amer. J. Med., 1955, **19**, 4.
12. Florey, Sir Howard, Ed.: General Pathology 2nd edition, W. B. Saunders, Co., 1958.
13. Williams, Dennis, Ed.: Modern Trends in Neurology, Butterworth & Co., London, 1957.

POST-GRADUATE MEDICAL TRAINING IN ENGLAND

BEVERLEY CLARK BURGESS, M.D., 5T4

HAVE you heard of all the opportunities available in England for post-graduate medical education? They are almost unlimited.

Whether you take a year or two here, right after internship or much later in your training (it is best to get at least a good first year internship before coming to England and have saved a little cash), your scope is broadened both personally and professionally and it stands you in good stead for the rest of your practising life. The training is approved toward our post-graduate examinations; the Canadian examining bodies accept any hospital appointments or training program which the English Royal Colleges approve, and vice versa for our Canadian degrees and internships in qualifying for English jobs and exams. Post-graduate medical education here has been developed into a fine art, is highly organized and beautifully executed in every detail. London is swarming with doctors from all over the world, chiefly from Commonwealth countries, and Canadians, though very much in the minority, are given an especially hearty welcome.

It is important to get training arranged well in advance and this is done by sending details of previous training, proposed course of study and final aim with a reference if possible from your dean of medicine or from someone with a personal contact (introductions are much more important in the Old World than in the New), to Professor Sir Francis Fraser, British Postgraduate Medical Federation, 18 Guilford St., London, W.C.1., England. This Federation has all the necessary contacts and, if you wish, will supervise your training program from start to finish in an expert and friendly manner, hold references for you during appointment hunting and send reports back to your medical school and to any examining body designated by you. Their advice is invaluable. Further information may be gained from the *Lancet*, August 30, 1958, or the B.M.J. of September 6, 1958, or from the Handbook of the Federation.

If you wish to arrange a place to live ahead of time, London House, Guilford Street, London, W.C.1., and William Goodenough House, Mecklenburgh Square, London, W.C.1., are central and intended for Commonwealth post-graduate students in London. There are many pros and cons to living in such residences but for Canadians two facts stand out: wherever you live in England there are few modern conveniences, and these residences are clean and centrally heated!

One gets caught up in exam fever over here and the courses for final F.R.C.S. in the various subjects and for medical degrees are excellent. Fees are about $80.00 for ten to twelve weeks. The main courses taken are the Neurology course at Queen Square, the Cardiology course at the Heart Hospital on Wimpole Street, the Chest course at the Brompton Hospital and the general medical course offered biannually by Hammersmith Postgraduate Medical School. Most places are reserved for non-English doctors. A typical day for instance at the Hammersmith course starts with a 9 o'clock lecture, 10 o'clock clinical demonstration, 11 o'clock out-patient session. In the afternoons clinics and special lectures are held.

It is possible to get a London or Edinburgh F.R.C.S. after your name three

years from graduation instead of five or six as at home, (though this is seldom attempted or achieved). I doubt whether I should be giving out this information! It is easy to get Canadian hospital appointments with these degrees but to make them permanent and to practise a specialty in most cases in Canada it is necessary also to pass the certification exams of the Canadian College. For the M,R.C.P. (pass rate 8–10%) it is recommended to do at least one hospital job in England to get up to the required standard, but M.R.C.P. is not recognized in Canada so most people take the courses and go back home ready to write exams.

It is not easy to get an English teaching hospital post and the plum jobs are reserved for those on travelling fellowships or backed by their university or very advanced in their specialty. It is possible often to get taught just as well in non-teaching posts here but appointments are awarded only after advertisement and interview, and cannot be arranged from the other side of the Atlantic very well. There are a group of "fringe" hospitals who have on their staff consultants from teaching hospitals and sometimes working for one of these men in a B hospital leads to a good teaching hospital post. You get better jobs if you first do a course and get a degree, e.g. the Primary F.R.C.S. for surgical jobs (pass rate is only 11–20% and competition keen). There are a greater number of surgical jobs offering much more operative experience and at an earlier stage in training in England than Canada. At Guy's Medical School in London they offer an unpaid three-month studentship for Commonwealth doctors who are taken on as part of a surgical team and are an integral part of the hospital though they do no operating.

After a year of rotating internship, first job salaries here start at £575 a year less £125 living costs if you live in, and on this basis take-home pay for a single man is about the equivalent of $17.00 Canadian a week. Living in London costs about the same as Toronto and about 25% less in other parts of England. Senior House Officer posts available after 12–18 months from graduation pay £825 per annum gross ($2.70 to the sterling pound) and registrarships following this pay from £920 gross with £175 deduction if you live in hospital, so that take-home pay is about £10 11*s*. weekly. Taxes are very high. Several Canadian graduates at present have plum jobs in England: one with a Canadian F.R.C.S. has a house job at the Sick Children's Hospital at Great Ormond Street; a 5T6 girl is on the house at the Maudsley Psychiatric Hospital and a 5T1 man has a registrarship in the chest unit at Hammersmith. Under the National Health, specialist services are highly organized and as such there is a lot of work in specialist posts. A doctor here is a public servant and has to work pretty hard for his money. It would be a good thing if more Canadian doctors could work for a while in a National Health post and decide first hand what system is best. Among doctors here enthusiasm for the system seems to be waning and dissatisfaction growing.

Under the post-graduate medical federation are the P.G. Medical School of London with its international reputation for medical research and a number of Institutes which regularly hold courses —Institutes of Cancer Research, Dental Surgery, Laryngology and Otology, Obstetrics and Gynaecology, Orthopaedics, Psychiatry, Urology and Dermatology, to name only a few. Whatever your interest, it will be served. The Institute of Basic Sciences holds courses twice a year for people studying for higher degrees or for those wishing to broaden their general knowledge and they afford

opportunities for research to specially selected students as do all the other institutes. The Institute of Cardiology holds courses of instruction both part and full time, and has Dr. Paul Wood on the teaching staff; such men are an inspiration to listen to, watch and work with, and often impart brand new concepts which create new interest for the doctor and at any stage this can be helpful. It is impossible to outgrow what London has to offer in specialist training. The Institute of Child Health is situated at the Hospital for Sick Children, Great Ormond Street. They offer a ten-week course in Paediatrics with splendid clinical material which is a boon to the general practitioner, internist and paediatrician alike. The Institute of Diseases of the Chest makes use of the clinical facilities of the wards and extensive out-patient departments of the Brompton Hospital and of the London Chest Hospital and has on the staff Sir Russell Brock. For doctors advanced in their specialty there are registrarships and unpaid assistantships (often on research lines) with such men, as there are in most of the institutes.

The Institute of Neurology at Queen Square, home of Sir Francis Walsh, Gowers and Jackson of Jacksonian epilepsy fame, offers teaching and ward rounds on a very high plane conducted by top neurologists: Sir Russell Brain, Macdonald Critchely, E. A. Carmichael, S. P. Meadows, Keith Lyle and others. Their teaching is invaluable to the internist or neurologist and they offer courses elementary and advanced. They have only referred patients and are drawing from most of 40 million people, plus baffling cases from other countries; the clinical material is unbelievable and almost every out-patient teaching session is a revelation.

The Institute of Ophthalmology offers research facilities, elementary and advanced courses, good library (present in most of these institutes), pathology museum and out-patient departments of Moorfields eye hospital where they see up to 500 patients each morning. We in Canada are unacquainted with the volume of clinical material available in a single city whose population is greater than half the population of Canada. The farthest city in England is only about six hours distant by train; thus London hospitals are able to drain a very large population.

Living in London is in itself an unforgettable experience. The people you meet from all over the world and the very different way of living are a part of life which you'll remember vividly, aside from the golden educational opportunities. And the continent is so close that taking holidays there is an easy matter compared with having to travel three thousand miles before touching those glamorous shores. On the continent you will enjoy visiting well-known medical centres such as Zurich and you will find that as a Canadian you are welcome wherever you go. It is possible to get jobs in the continental centres, either locums or permanent appointments, especially if you know their language. The American Hospital in Paris takes locums frequently (at $1 a day plus room and board) and living in Paris can be a lot of fun too.

You'll never regret time spent "over here".

THE MANAGEMENT OF BENIGN ANO-RECTAL DISEASE

HARVEY NEWTON, 5T9

ONE of the most ancient and most common conditions that attacks the human race is rectal disease. Some seventy-five percent of people will suffer with some form of rectal disease during their lives.

This most brief summary will deal primarily with management of these conditions although a brief description of the lesion and its important features is included.

CLASSIFICATION OF BENIGN ANO-RECTAL DISEASE:

A. CONGENITAL:

1. *Stenosis, Imperforate anus, Sexual fistulae.*
2. *Pilonidal Sinus.*

B. ACQUIRED:

1. *Inflammatory*:
   - (a) Cryptitis
   - (b) Proctitis
   - (c) Pruritis ani or perianal dermatitis
   - (d) Abscesses
   - (e) Fistula in ano.
2. *Vascular*:
   - (a) External hemorrhoids
   - (b) Internal hemorrhoids
3. *Mechanical*:
   - (a) Fissure in ano.
   - (b) Rectal prolapse
4. *Traumatic*:
   - (a) Operative
   - (b) Due to injury
   - (c) Strictures
5. *Benign Tumours*:
   - (a) Adenoma
   - (b) Anal warts.

A. CONGENITAL:

1. *Stenosis*: A narrowing at the junction of the rectum and anus.
   Imperforate anus: The hindgut fails to canalize.
   Sexual fistulae: The hindgut may extend into the urethra, bladder, vagina or uterus.

Treatment: Stenosis is handled by rectal dilatation. Imperforate anus: The upper blind pouch is surgically brought down and sutured to the anal skin. Nasogastric suction and blood transfusions are urgently required. Sexual fistulae: Surgical intervention is required to relieve the obstruction.

2. *Pilonidal Sinus*: In this condition there has been faulty coalescence of skin in the sacral-coccygeal region. A small skin-lined tract is thereby formed. This condition causes no trouble unless the tract becomes infected.

Treatment: In the acute stage the tract should be drained and in a quiescent period, excised, destroying epithelium and allowing for healing with granulation tissue.

B. ACQUIRED:

1. *Inflammatory*:

(a) Cryptitis—Papillitis. In this condition the crypts of Morgagni become inflamed as they collect infected fecal material. Fecal material thus harbours the pathological organisms and traumatises the crypts.

Treatment: Hot sitz baths and dilation of the sphincter are the best early measures while surgery frequently becomes necessary at a later stage. The free edge of the crypt is cut for drainage.

(b) Proctitis: This is a broad term to define any inflammation within the rectal mucosa. The etiology is long and varied and includes allergy, vitamin deficiency, endocrine dysfunction, chronic constipation, purgatives, rectal injections and prolapsing hemorrhoids which are the non-pyogenic causes. The specific organisms include the dysenteries, T.B., gonococcus, syphilis and lymphogranuloma venereum.

Sigmoidoscopic and pathological assistance are required to establish a cause.

Treatment: Bed rest, oil enemas and bland diet will cure most cases of proctitis. In cases of non-specific obstinate proctitis, sulphguanidine may arrest the disease. Penicillin is used for gonococcal and syphilitic afflictions while streptomycin is specific for lymphogranuloma venereum. Surgery is only indicated for complications such as anal fissure and acute anal hematoma.

(c) Pruritis Ani: This condition refers to local irritation about the perianal skin and is readily amenable to treatment.

Eight clinical varieties are recognized:

(i) Lack of cleanliness,
(ii) Excessive sweating,
(iii) Mucoid leak,
(iv) Abnormal anal moisture,
(v) Parasitic causes,
(vi) Allergy,
(vii) Mycotic infections,
(viii) Idiopathic pruritis.

Treatment: Hygiene is essential. The anal region should be cleansed, dried and powdered. The stool should be kept soft with milk of magnesia. Antihelminthics, antihistaminics and antifungal drugs are employed where indicated. In severe idiopathic varieties, phenol and alcohol injections to the perianal regions will often relieve the condition.

Surgery is used when all else fails and the symptoms are severe. A wide excision of skin in a butterfly shape with healing by granulation tissue is employed.

(d) Abscesses: Abscesses occur as localized collections of pus outside the muscle layers of the rectum above or below the levator ani. Streptococci and B. coli are the frequent offenders. Abscesses are prone to fistula formation.

Four clinical varieties are described:
(i) Perianal abscess: Results from infected crypts, hair follicles or sweat glands.
(ii) Submucous abscesses are found in the lateral walls of the rectum.
(iii) Ischio-rectal abscesses are found in the ischio-rectal fossa and result from blood stream or rectal infection.
(iv) Pelvi-rectal abscesses localize above the levator ani and often complicate an abdominal infection. They are serious but fortunately very rare.

Treatment: Perianal and submucous abscesses require early incision, drainage and packing, with sitz baths post-operatively. Ischio-rectal abscesses are drained with a T-shaped incision, saucerized and may be packed. Pelvi-rectal abscesses are drained through the ischio-rectal fossa.

(e) Fistula in Ano: A fistula results most commonly from a rectal abscess where a sinus lined with granulation tissue connects internally to the anus and rectum or externally to perianal skin, or both.

A fistulous opening in the perianal skin from which pus exudes is highly suggestive. Pyogenic and tuberculous organisms are the common offenders. A probe may be passed to outline the tract.

Treatment: Operative management is the only certain cure. The main tract with any secondaries, is laid open over a probe and a dressing applied. The wound must be rechecked for pus which may indicate that a secondary is still present and surgery may have to be repeated or silver nitrate applied. Soft stools are necessary for healing.

2. *Vascular (Venous) Lesions*:

(a) External Hemorrhoids: The external hemorrhoidal plexus, which surrounds the lumen of the anal canal and empties into the systemic circulation via the inferior and middle hemorrhoidal veins, may become dilated. An acute attack results from straining and a hematoma forms due to rupture of a dilated vein.

Treatment: In the acute stage the hematoma should be excised under local anaesthetic. Massive thrombosis should be treated with firm compresses every three to four hours and stools should be kept soft. In chronic external hemorrhoids, excision under local anaesthetic with granulation healing is the method. Palliative therapy is recommended for small or inflamed piles.

(b) Internal Hemorrhoids: The internal (superior) hemorrhoidal plexus is a network of veins located above the ano-rectal line. These veins are drained by the superior hemorrhoidal vein and into the portal circulation. Dilatations of the internal hemorrhoidal plexus occurs at 3, 7 and 11 o'clock, with secondaries between them. Complications such as hemorrhage, strangulation and suppuration are common in this condition. Diagnosis requires proctoscopic examination.

Classification:

1st degree: blood but no prolapse.
2nd degree: prolapse, but reduce themselves.
3rd degree: prolapse, but require digital replacement.
4th degree: prolapse, but cannot be replaced.

Treatment: Palliative for 1st and 2nd degree internal hemorrhoids. Liquid paraffin, sitz baths and bed rest will control this variety. Thrombosis, prolapse and infection require firm compression. Five cc. of phenol in almond oil, injected near the base of the hemorrhoid at weekly intervals may result in cure in early stages. Surgery is recommended for 3rd and 4th degree piles where the base of the pile is ligated and bleeding points carefully tied off.

3. *Mechanical*

(a) Fissure in ano: A fissure occurs as an elongated slit-shaped ulcer usually found posteriorly and beginning near the ano-rectal line and extending downward to the anal opening. The condition is initiated by a tear from a hard fecal mass and is made worse by repeated constipation. A fissure is often accompanied by a hypertrophied tag of anal tissue known as a sentinel pile.

Treatment: Bed rest, mineral oil and sitz baths will greatly alleviate the suffering. Injection of 10 cc. of 5% proctocaine into the sphincter will relieve the pain due to muscle spasm. Bland diet, hot compresses and suppositories will further relieve the suffering. Cauterization of the fissure is also widely practised with rewarding results. Surgery is advocated for chronic fissures where all other means have failed and the pain is intolerable.

(b) Rectal Prolapse: Two types of prolapse occur: partial, (mucous membrane only), and complete (entire thickness of rectal wall). Partial prolapse is common in the very young while complete prolapse is not common and affects the older age group. Complete prolapse is in effect a sliding hernia of the pouch of Douglas through a weak levator ani.

Treatment: Babies are handled conservatively and the mother is directed to replace the prolapse and closely supervise the child so that it does not strain. Linear cauterization or phenol injection can be used if meticulous efforts of the mother fail. The stools should be kept soft. In cases of complete prolapse, surgical repair should be instituted. It is treated like a hernia elsewhere by re-

construction of the pelvic floor thereby preventing prolapse from above.

4. *Traumatic Injuries of the Rectum*:

(a) Operative: During operation on the urethra, prostate, vagina or uterus, perforation of the rectum may accidentally occur. Injudicious use of sigmoidoscopy or enema nozzles may result in the same injury. Immediate repair is indicated and usually with an associated colostomy.

(b) Trauma: Perforation of the rectum may result from crush injuries or the introduction of foreign bodies to the rectum. Treatment centres around removal of any objects and checking the wall under anaesthetic. If a perforation is discovered a colostomy may be performed until the perforation heals.

(c) Strictures: Strictures occur after operation, perforation, radiation and lymphogranuloma inguinale, resulting in fibrous stricture formation. The result is difficulty in defecation, straining at stool and ribbon-like stools.

Treatment: This revolves around dilatation with rectal bougies of graduated size. Operation may be deemed necessary in which case internal proctotomy, left sided colostomy or perhaps abdominal perineal resection may have to be performed.

5. *Benign Tumours*:

(a) Adenoma: The commonest benign tumour is a bright red adenoma or simple polyp. Other tumours like lipomata, fibromata, mymomata, lymphomata and angiomata are rarities and need only be mentioned.

The adenoma is the size of a pea which may enlarge to 3 cm., become sessile and pedunculated and may be the forerunner of malignancy. Rectal polyps are usually multiple—two or more—and this is termed polyposis, (not to be confused with multiple polyposis). Rectal pressure, fecal trauma, erosion, ulceration or impaired blood supply cause scar tissue formation and eventually more fibrosis. Diagnosis is easily made with the sigmoidoscope.

Treatment: The polyp is grasped by forceps and snare or cauterized. This condition is premalignant and surgery is the only logical way to manage it. Diathermy is particularly applicable to this condition.

(b) Anal warts or multiple papillomata of the skin do occur and are removed with scissors under local anaesthetic and packed with petroleum gauze.

Acknowledgment

I should like to thank Dr. F. Mills for his kindness in reading and criticizing this paper.

Bibliography:

1. Gabriel, W. B.: The principles and Practice of Rectal Surgery, Ed. 4, 1948.
2. Moseley, H. F.: Textbook of Surgery, 1957, pp. 486–499.
3. Clinical Review of Common Rectal Disorders, William R. Warner Drug Co.
4. Milligan, E. T. C.: Proc. Roy. Soc. Med., 1943, 36, 365.
5. Bacon, H. E.: Anus, Rectum and Sigmoid Colon.
6. Hamilton Bailey: Physical Signs in Clinical Surgery, pp. 249.

MEDICINE MADE EASY

P. C. F. MOORE, 5T9

I HAVE just finished a grand tour of inspection of a sewer plant.

This event marks a new epoch in my life for now my education is complete. Years of study have acquainted me with the laws of physics and chemistry in all their baffling intricacies; the dynamics of life, studied from the simple amoeba through to the complex homo sapiens, is no secret now to me; into the realm of metaphysics I have delved to emerge a master of abstruse philosophy and my intimacy with the better writers of these and past times has broadened and ennobled my scope. Only one phase of my learning had suffered neglect—and the tour of the sewer plant was the remedy.

Having thus attained this exalted state, my purpose holds to share with others certain jewels of my wisdom. It is into the mysteries of the study of medicine that I choose to shed my light.

Passage through medical school is an opportunity unique among the experiences of men to display the utmost in subtlety, ingenuity and skill. Woe unto that neophyte who seeks to gain our ranks possessing not these qualities, and misery unto him who, having entered our portals, fails to exploit them to their fullest. But apart from these essentials, there are certain tactics, known only to a favoured few, the knowledge of which renders medical school an easy task; these I plan to divulge, to revolutionize its methods forever.

A consideration of the premedical years forms only the most minor part in this discussion, for although I am the last to belittle their efficacy—or indeed to label them easy—they are definitely limited with respect to possibilities for exhibiting the diabolical shrewdness with which this article intends to deal. Suffice it to say that he who can master Millikan's oil drop experiment and the Wheatstone bridge, adjudge the proper moment to throw pennies and who remembers to focus up and down will have little difficulty in grasping the more significant concepts.

Likewise, the first medical year is restricted as to the opportunities to develop the particular talent to which this article is dedicated, although the student who consistently switches the labels during an anatomy bell-ringer is undeniably endowed with an aptitude for craftiness and subterfuge.

But it is to the clinical years that our attention is directed; here lies the meat of our quest. Here every day provides a challenge and with every hurdle successfully leapt we are nearer to the perfection of our art.

The clinician is among the most outstanding of our problems. To deal with him effectively we must summon every resource. Flattery and cajolery are child's play; knowledge of *everything* is difficult; but the real key to success is to mingle humour and camaraderie, poise and dexterity, slyness and good judgment to the end that you have his respect and admiration. Skillfully employed, these means can have the clinician eating from your hand; *he* will laugh at *your* jokes, to him your presentation of a patient will be enviable, and as you go on rounds he will pat you on the back and take your arm.

To illustrate, I shall enumerate some of the techniques to which no small number of students owe their success.

The first to come to mind is the use of what is known in some circles at least, as

rumballism. The object here is to convey the impression of vast knoweldge when confronted with a problem about which you know nothing. On several occasions I have been privileged to observe this technique and while as yet I have not developed it into a fine art, it has shown by the test of time to be of value. One raises one's eyebrows superciliously. This immediately suggests that the problem is simple and completely within understanding. Then the mouth is opened and with authority a quick succession of words is poured forth, bearing a repetition of the question asked and a few catch pharses that apply to any problem. The remainder of the words, while not intelligible on close analysis, are delivered with such speed and assurance that the clinician can only nod agreement and raise his eyebrows too.

Another technique is that of classification. This is not the ordinary type where one begins with things congenital and acquired, but one in which, when confronted with the problem of a diagnosis, a set number of conditions, the possible findings in which are countless, are at one's finger tips. I know a student who dealt with all neurological disorders quite convincingly by suggesting that they were manifestations of either a ruptured berry aneurysm or tabes dorsalis.

Quick thinking cannot, of course, be over-emphasized and this ability, though possessed by only a few, can establish one on the road to fame. Picture the look of humble respect on the face of the clinician who, after having demanded the means of preventing spread of gonorrheal conjunctivitis from the right eye to the left, received the unflinching answer: "contraception."

Certain students are handicapped by being able, after exhaustive study, only to remember details of the utmost triviality. They should not despair, for even these can be put to use in our scheme. Think of the possibilities of being able to recite that the clavicle is thrice unique, and then there is Boyd's classic description of the cretin, capable of impressing even the least emotional clinician: "What was intended to be created in the image of God has become what has been called the pariah of Nature, and all for want of a little iodine." And of course the student who is familiar with the Laurence-Moon-Biedl syndrome or who knows that pseudohypertrophic pulmonary osteoarthropathy is the same thing as clubbing of the fingers is at a decided advantage though he remembers little else.

Now when we come to techniques used in oral examinations we are dealing with a speciality so advanced that the development of proficiency is within the reach of only a gifted few. Two of the subtler approaches are (a) hesitation and (b) sympathy.

The hesitation method is as follows: You have been asked, for example, as a prelude to further discussion, the differential diagnosis of acute appendicitis. You manage to run through the various conditions ranging from acute salpingitis to renal colic. Getting towards the bottom of the list, you stumble over mesenteric lymphadenitis, perhaps mispronouncing it, or at any rate mumbling it questioningly. Immediately your examiner pounces on you demanding a thorough description of this condition, and of course he has fallen right into your trap. You proceed, timid at first, but developing more confidence as you go, to give him the list of signs and symptoms, pathology and treatment, having completely familiarized yourself with the condition beforehand. By these means, the examination is conducted along lines in which you are well versed.

The sympathy act is quite touchy and only to be used in the most desperate circumstances by those of great stamina. For my own part, I used this technique

only once and reached a peak of such brilliance that I can never hope to match it again. The case to which I refer was a surgery oral and things were going badly. My answers were becoming less and less satisfactory to the examiner when I hit upon a scheme that saved the day. First I gave what would appear to be a sign of defeat by mopping my brow, and then, taking a deep inspiration, I managed to pop the button off my coat. Wobbling at the bedside, appearing all but paralysed, it seemed as if that button were the last of me to fall apart. I continued to mumble on, taking no notice, keeping up a brave, if pitiable front. It was too much for the examiner. He gently retrieved my button, put it in my pocket and dismissed me with a soothing, fatherly kindness. As I say, this method requires great stamina.

Having dealt with clinicians and oral examinations, the two main obstacles in the course of one's progress, I shall continue with a brief discourse on some of the minor points, which though of less consequence, are worthy of our consideration.

Hypochondriasis, of course, is one of the pitfalls of medical students and while to be avoided if one is to maintain a fair degree of sanity, a healthy smattering of it will at least stimulate the student to make a thorough investigation into the diseases from which he is suffering. I know of one student who exhibited the symptoms at least, if not the signs, of amoebiasis, Marie-Strumpell disease, bronchogenic carcinoma, irreducible hemorrhoids, status asthmaticus and housemaid's knee all in the course of his second medical year. In due time, he was an authority on each of these subjects and I understand that he continues to augment his knowledge this way every year.

Being a "character" is to some medical students an indispensable agency to the development of self-confidence. Among the tactics used in this respect are wearing the sleeves of your lab coat rolled above the elbows, growing a beard, calling clinicians by their first names, writing histories in Latin, being a grandmother and standing first in the class. Other means will no doubt occur to the ingenious.

Nurses can be a source of inspiration and education. To improve public relations with them, volunteer to do their hemoglobins, as they place great emphasis upon this. Having established yourself, you can learn a great number of useful procedures, such as taking blood, doing lumbar punctures, inserting stomach tubes, and many others. I once went through four sterile catheter trays in an attempt to relieve a woman's bladder distension and had a review of anatomy from five different nurses at the same time. Ignorance of anatomy can be rather provoking.

One-upmanship is a demoralizing technique which of late has fallen into the hands of rank amateurs, but for the sake of completeness, a brief example will be described. One of your classmates has just finished a brilliant and comprehensive dissertation of the causative factors in myeloid leukemia. You blandly add, after his proud finish, that Stanislowski, a promising young Russian scientist has just published a paper in the Siamese Medical Journal postulating a new globulin, Z-260, the absence of which has caused myeloid leukemia in six generations of three-toes sloths, and your astounded friend is reduced to an amorphous mass.

It will be seen that the possibilities for inventiveness are legion and that no more need fears, qualms or inhibitions plague the medical student. With practice, even the most limited student can sail through his courses with a new perspective, a new fervour and a new hope. A word of caution, however, is in order:

ignominy and disaster await the student who fails to execute these principles with anything but the ultimate in cunning and deftness and those who doubt their ability herein must conform to the old school and labour after knowledge with endurance.

So saying, my labours with endurance I resume!

CASE REPORT

GLORIA PIERCE, 5T9

THIS was a very interesting rare case I was fortunate to have during a summer internship in Santa Barbara, California.

The patient was a 69 year old white widow, living alone, 5′ 4″, 120 lb. Her first admission was on November 25th, 1957. At this time her chief complaint was a vague discomfort in RLQ of abdomen for the previous four months. Also she had noticed a mass in the RLQ for five weeks.

History of Present Illness:

In August she noticed excessive gaseousness and a sensitive spot in the RLQ when gas was passed. Also she had some indigestion—eructation on several occasions. At no time did she have any pain of any consequence. There was no change in bowel habits or the stool; there was no melena or bleeding by rectum. She first noticed a lump in the RLQ of her abdomen about 5 weeks ago and this had grown considerably in that time.

The rest of the history was non-contributory except that she had extreme weakness on standing and a weight loss of 5 lb. in the last 4 weeks although her appetite and eating habits were unchanged.

Physical Examination:

She presented as a white female who looked her stated age and was lying quietly, not appearing acutely ill but with a very pale colour. Her pertinent positive findings were pale conjuntivae and buccal mucosa. Also she had a soft systolic murmur at the apex and the apex beat was 12 cm. from the midline in the 6th interspace. The B.P. was 178/90 and pulse regular at 88 per minute. Her liver was palpable 2 fingers-breaths below the right costal margin, but the edge was soft and no irregularities were palpable on the surface. In the RLQ there was a large, 8 × 10 cm. hard, slightly movable mass which was irregular in surface and circumference and only slightly tender to deep palpation. Also much borborygmi were present. Rectal examination was normal.

Laboratory Findings:

A Haemoglobin of 6.2 Gm %, a RBC of 2.7 m, a Hematocrit of 23 vol. %, and a WBC of 7,750. Her smear showed marked microcytic hypochromic anaemia. Urinalysis was normal.

Provisional Diagnosis:

Adenocarcinoma of the caecum, probably far advanced, but perhaps resectable. This was substantiated by GI series, showing a large irregular filling defect in the caecum and lower ascending colon.

Before operation she was given 8 blood transfusions, raising the Hb to 14.2 Gm % and the haematocrit to 45. Routine preoperative bowel prep was done. At operation a RLQ transverse incision

was made and the upper caecum and lower ascending colon were found to be the site of a tumour measuring approximately 10 × 8 cm., the size of a medium grapefruit. The mass was hard, nodular, and extended through to the serosa of the bowel but had no apparent other extension. The liver was normal to palpation except for adhesions to a small fibrous gallbladder. The spleen, kidneys, and rest of the pelvis were normal to palpation. The caecum was mobilized and the right colic and ileocolic arteries tied. The colon was sectioned at the proximal end of the transverse colon and the end turned in. The ileum was transected approximately 8 cm. from the ileocaecal valve. An ileotransverse colostomy was performed. She withstood the operation well and her postoperative recovery was excellent.

Pathological Report:

The caecum and ileocaecal valve were normal and no neoplastic mesenteric nodes were present. The neoplasm in the posterior wall of the ascending colon opposite the ileocaecal valve was bulky, mottled tan red, and a homogeneous glistening ivory colour on cross-section. The microscopic sections showed monotonous sheets of plasma cells infiltrating the muscularis and subserous layers. Most of the cells were small mature plasma cells but considerable multinucleated cells and pleomorphism were present. Diagnosis was malignant plasmacytoma of the ascending colon.

Further lab tests were done at this time. No Bence-Jones protein was found in the urine. Serum electrophoresis showed subnormal albumin of 2.7 Gm %, although the total protein was 6 Gm %. Of the globulins, alpha 2 was 1.1 (the upper limit of normal in this lab being 0.9). However these findings are not significantly abnormal to be diagnostic of multiple myeloma.

Following the operation the patient had routine check-ups and continued in good health until June 9th, 1958 when a hard round mass was found just above the pubis to the right of the midline, slightly moveable and slightly tender. Bimanual pelvic examination revealed the mass apparently attached to or growing from the right corner of the uterus. Moving the cervix moved the mass and it was thought to have the feeling of a uterine fibroid. No anaemia was present and no gastro-intestinal symptoms were present.

An exploratory laparotomy was performed on June 13th. Loops of small bowel were found to be adherent to a firm irregular 6 × 7 cm. mass, thought to be at the site of the old anastomosis, although the pathology report later showed the origin to be 6 cm. proximal to it. Two loops of small bowel and the old colostomy had to be excised, and two end-to-end small intestine anastomoses and an end-to-end ileotransverse colostomy were performed. Post-operative course was uncomplicated and the patient was discharged in good condition, to be carefully followed at 3-monthly intervals.

The diagnosis from the pathology report was recurrent malignant plasmacytoma, this time showing hyperplasia in the mesenteric lymph nodes.

BOOK REVIEW

VARIED OPERATIONS—Herbert A. Bruce

Varied Operations is the autobiography of Dr. Herbert Bruce and is a vivid summary of ninety years of his happy and successful life. Dr. Bruce studied medicine in Toronto before the turn of the century and later became one of this city's most brilliant surgeons.

Among other accomplishments of his career, he distinguished himself in World War I, became Lieutenant-Governor of Ontario and was later a member of Parliament. Frequently involved in controversial issues, he fought for many causes, and his opinions on the Canadian Medical Services in World War I, on the radical measures of Mitchell Hepburn, on Mackenzie King's handling of problems in World War II, on Christian Science, on birth control and sterilization are presented with positive zest.

His story flows with many absorbing anecdotes from his active life and he introduces us to a great number of famous people—of Toronto and of world renown with whom he came in contact. He presents an interesting history of Toronto's development along with the emergence of our Faculty of Medicine; the activities and circumstances of our early hospitals are portrayed and the student of today will enjoy the comparison. Some bizarre and particularly delicate operations are described and some choice experiences from his own practice are related. His life as Lieutenant-Governor and as a Member of Parliament gives an informative and glamourous look into the non-medical side of his career, showing him to have displayed great courage and leadership.

This book is delightfully written with charm and energy and to the medical student and to the doctor should serve not only as a source of pleasure, but of inspiration to greater achievement.

There is a Foreword by the Rt. Hon. John Diefenbaker, P.C., Prime Minister of Canada.

Published by Longmans, Green and Co., price $6.00. P.C.F.M.

NON-NARCOTIC COUGH SPECIFIC

to control the cough
and calm the patient . . .

Expectorant action
Antitussive action
Antihistaminic action
Sedative action

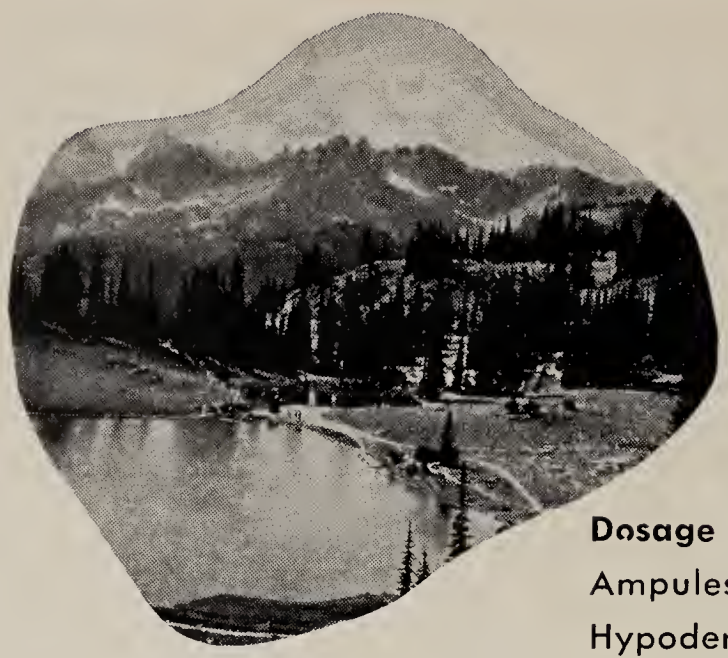
Promatussin

EXPECTORANT
Promethazine Hydrochloride
with Dextromethorphan

Wyeth
Registered Trademark
WALKERVILLE, ONTARIO
MONCTON · MONTREAL
WINNIPEG · VANCOUVER

designed for performance
THROUGH THE DAY
AND
THROUGH THE NIGHT

for the peak of analgesic efficiency



DILAUDID ®

brand of DIHYDROMORPHINONE

Dosage Forms of Dilaudid hydrochloride:

Ampules: 1 cc., 2 mg. each.

Hypodermic Tablets: 2 and 4 mg. each.

Oral Tablets: 2.7 mg. each.

Multiple Dose Vial: 10 cc., 2 mg. Dilaudid sulfate per cc.

Dilaudid, Trademark reg., marketed by Knoll Pharmaceutical Company, Orange, N. J.

MERCK & CO. Limited *exclusive distributor*

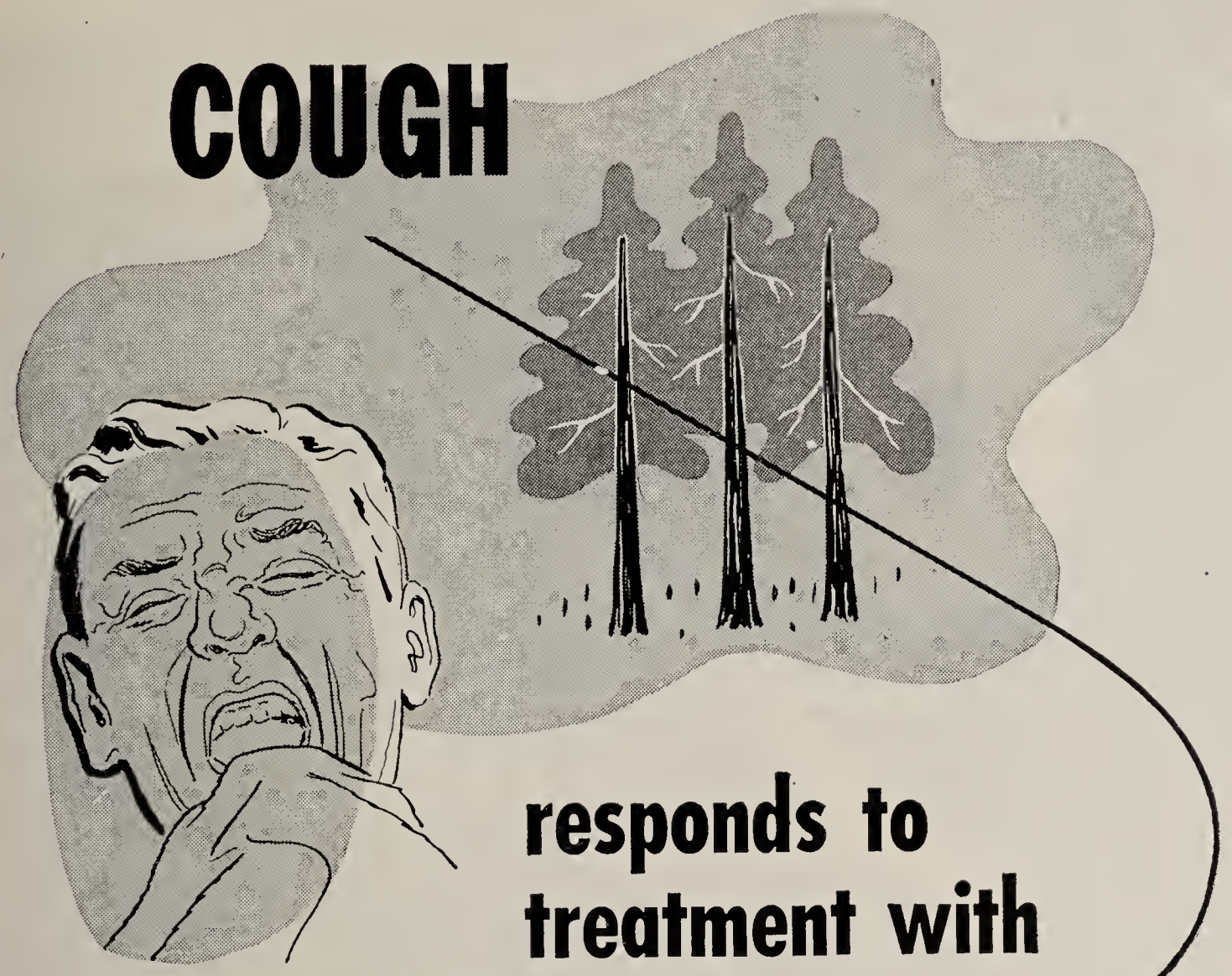
MONTREAL TORONTO VANCOUVER VALLEYFIELD

Homewood Sanitarium

A private hospital for the diagnosis, care and treatment of all types of psychiatric disorders. An adequate staff of specialists study each patient carefully and fit the treatment to his individual needs. Excellent cuisine, comfortable accommodation and personal attention of specially trained nursing staff contribute to the recovery of the patient. All types of modern therapy are available, including Psychotherapy, Insulin and Electro-shock, combined with excellent Occupational and Recreational facilities. The Sanitarium buildings, accommodating 225 patients, are situated in a very attractive setting within the confines of the City of Guelph. Patients who are policyholders with the Ontario Blue Cross Plan for Hospital care or Life Insurance Hospitalization schemes are eligible for benefits. Visits by physicians and other interested parties are always welcomed. For further information write or telephone (TA. 4–1010).

A. L. MacKINNON, M.B., Medical Supt.

THE HOMEWOOD SANITARIUM OF GUELPH, ONTARIO, LIMITED



COUGH

responds to treatment with

"PINOCODEINE" (N)

BRAND

Each fluid ounce contains:

Pinus strobus	32 gr. (2.1 G.)
Prunus virginiana	32 gr. (2.1 G.)
Sanguinaria canadensis	4 gr. (0.25 G.)
Populus balsamifera	2 gr. (0.12 G.)
Chloroformum	1 min. (0.06 cc.)
Codeinae phosphas	1 gr. (60 mg.)

(N) *Telephone narcotic prescription permitted.*

DOSAGE: One or two teaspoonfuls every four hours.

Packaged in 16 fluid ounce bottles.

Frosst

Charles E. Frosst & Co.

MONTREAL CANADA

ROVAMYCINE PEDIATRIC

Oral suspension containing 125 mg. of Spiramycine base per 5 ml. (teaspoonful) in a pleasantly flavoured combination.

a safe oral antibiotic

ROVAMYCINE

SPIRAMYCINE

- RESPIRATORY-TRACT INFECTIONS
- STAPHYLOCOCCAL INFECTIONS
- RESISTANT CASES TO OTHER ANTIBIOTICS
- WHOOPING COUGH
- FURONCULOSIS, PYODERMATITIS

Capsule of 250 mg.

direct activity against GRAM-POSITIVE organisms

remarkably well tolerated

information upon request

Poulenc *Limited*

8580 Esplanade, Montreal

a safe
non-narcotic
cough suppressant
in **solid** form

Utilizing Becantex*, a unique antitussive 1½ times as effective as codeine, Tussets suppress coughs of various origin by dampening the cough reflex. Their systemic action is certain, rapid, and sustained via the cough center. Safe in children and the elderly, Tussets are efficient whether sucked, chewed, or swallowed. Relieve useless, irritating cough in colds, bronchitis, and heavy smoking, etc. Also most useful in whooping cough, and coughing associated with pulmonary tuberculosis, bronchiectasis, and pulmonary neoplasms.

average dosage:

e Tusset per hour, or as re-
ired. Packaged in 12's and
0's. *Sodium di-tertiary butyl
phthalene sulfonate, 15 mg.
r Tusset.

tussets

systemically effective antitussive

RANK W. HORNER LIMITED • MONTREAL

THE MONTREAL GENERAL HOSPITAL

(McGill University Teaching Hospital—720 Beds)

Applications are now being received for junior internship commencing June 16th, 1959. 48 posts are available (all these posts have been filled in the past five years). Preference given to Canadian graduates. Appointments are for one year but preference for senior internship in all specialties and in general practice given our own junior internes.

Rotations are on a two-month basis and include Medicine, Surgery, Obstetrics & Paediatrics. Honorarium is from $480.00 yearly to $630.00 yearly for single internes and from $960.00 yearly to $1,030.00 yearly for married internes plus full board, uniforms, laundry, as well as travelling expenses between the Montreal General Hospital and the Affiliated hospitals outside Montreal. Accommodation is provided if the interne is single.

If possible, an interview is desirable and arrangements should be made by writing to Dr. William Storrar, Medical Director, The Montreal General Hospital, Montreal, Quebec.

For further information, interne brochure and application form, please write to Dr. William Storrar, Medical Director, The Montreal General Hospital.